ASSESSMENT OF CAROTID INTIMAL MEDIA THICKNESS IN STROKE

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CERTIFICATE

This is to certify that the enclosed work **"ASSESSMENT OF CAROTID INTIMAL MEDIA THICKNESS IN STROKE"** submitted by Dr. N. KARUPPUSAMY. to the Tamilnadu Dr. M.G.R. Medical University is based on bonafide cases studied and analysed by the candidate at the Department of Medicine, Coimbatore Medical College Hospital during the period from January 2004-December 2005 under my guidance and supervision and the conclusions reached in this study are his own.

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DEAN

DECLARATION

I solemnly declare that the dissertation titled **"ASSESSMENT OF CAROTID INTIMAL MEDIA THICKNESS IN STROKE"** was done by me at Coimbatore Medical College Hospital during the period from January 2004 – December 2005 under the guidance and supervision of Professor Dr. P.JAMBULINGAM, M.D.

This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch – I) in General Medicine.

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CONTENTS

		Page No.
1.	INTRODUCTION	1
2.	AIM OF STUDY	4
3.	MATERIALS AND METHODS	5
4.	REVIEW OF LITERATURE	10
5.	OBSERVATION AND RESULTS	46
6.	DATA ANALYSIS	52
7.	DISCUSSION	61
8.	CONCLUSION	68
9.	BIBLIOGRAPHY	
10.	PROFORMA	
11.	ANNEXURES	

INTRODUCTION

INTRODUCTION

Stroke is one of the leading causes of morbidity and mortality and rank next to coronary artery disease and malignancy: Classically strokes are a disease of the elderly population with a peak incidence between sixth and eighth decades, but not uncommon in younger Age.

They cause ~ 200000 deaths each year in the united states and are a major cause of disability. Most cerebra vascular disease are manifested by the abrupt onset of a focal neurological deficit as if the patient was stuck by the hand of God.

The term cerebrovascular disease refer to any disease implicating one or more of the blood vessels in the pathological process such as abnormality of the vessel will occlusion by thrombus or embolism, rupture of a vessel, a failure of cerebral blood flow due to fall in blood pressure, a change in the caliber of lumen, altered permeability of vessel wall or Increased viscosity of blood.

Hospital based date revealed that 2% of all admission and 5% of Medical admission and around 25% of neurological admission due to Stroke.

According to a clinical survey conducted using records of the Harvard Co-operative stroke register in which the appropriate laboratory aids were used(CT-SCAN, four vessel angiography and CSF examination)cerebral information accounted for approximately 80% of the stroke a opposed to 10-15% due to intra cerebral bleed, Hemorrhage from ruptured aneurysm and vascular malformation constituted remaining,

The principal causes of cerebral infarction are

1) Atherosclerotic thrombus.
2) Systemic Hypertension.
3) Cerebral embolism.

The most common cause of cerebral infarction related to vascular disease is atherosclerosis which is of the most common disorder in adult human.

In this dissertation work a detailed study of the relationship of carotid Artery intima media thickness in the cerebra vascular disease and variable risk factor like Hypertension, Diabetes Mellitus, Smoking,

etc., causing thrombosis of blood vessel in the cerebro vascular disease.

An analysis of fifty adults with stroke between 20-60 year age admitted in the medical wards and neurology ward of our hospital.

AIM OF THE STUDY

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1).To study variable risk factor in cerebro vascular disease.

2). To study carotid intima media thickness in the cerebro vascular disease.

3).To study the different mode of clinical presentation in stroke in adults.

4). Correlative study of the above modalities.

MATERIALS OF METHOD

MATERIALS AND METHODS

The study was conducted in Coimbatore Medical College Hospital during the period of 2004-2005. The bed strength of this Hospital is 1020 and about 1000 patients are cared as inpatients, daily 4500 outpatients come to this hospital for treatment. This center is rendering medical services to a fairly large size of population nearly 10 lakhs, catering to the population in and around Coimbatore and nearby areas of Kerala including palghat. This being a post-graduate training center the fulltime services of a team of qualified neurologist and experienced medical personnel are available round the clock. This study was possible because of full cooperation and enthusiasm of various departments like Neurology, Radiology and with available facilities of this college.

The study includes fifty patients of stroke in patients admitted in medical wards and Neurology ward. A carefully elicited history and repeated clinical examinations were made use of in ascertaining the temporal profile of the disease and the probable area of brain that is affected.A detailed history taking done for risk factors like Smoking5

diabetes, Alcohol, Rheumatic valvular heart disease, Tuberculosis and family history of cerebrovascular disease.

A clinical search for extra-cranial carotid artery narrowing, cardiac diseases which can give rise to cerebral embolism, evidence for generalized arterial disease, systemic hypertension, diabetes mellitus, Rheumatic valvular heart disease and other risk factors were made.

Laboratory evaluation and other investigations

After detailed history and meticulous neuromedical examination including palpation and auscultation of carotid and peripheral pulses, the relevance and priority of any laboratory test in acute stroke should be at the clinician's judgment. The value of careful ophthalmoscopic examination of the retina and its vasculature for disease and embolic fragments needs emphasis.

1. Urine examination for albumin and sugar.
2. Estimation of blood sugar and serum lipid profile levels for detecting diabetes and hyperlipidemia which are predisposing causes of atherosclerosis.
3. Serological test for syphilis was done in all patients.

4. Hematological studies like complete hemogram with platelet count, ESR, bleeding / clotting and prothrombin time to find out conditions like polycythaemic, anemia, thrombocytopenia and bleeding disorders were undertaken.
5. When elevated ESR was present further test were made to identify the causes like systemic lupus erythematosus, sub acute bacterial endocarditis and tuberculous meningitis.
6. Anticardiolipin antibodies and lupus anticoagulant antibodies done for one patient.
7. Two dimensional echocardiography for detecting cardiac source of emboli.
8. ECG for detecting rhythm disorders was done.
9. CSF analysis was used to determine meningeal infection causing stroke and to confirm subarachnoid hemorrhage.
10. CT scan of brain was done to assess the lesion was infarction or hemorrhage and to locate the site of lesion.
11. Doppler study of carotid Artery intimal media thickness both internal and external carotid Artery on both side.

Significance of risk factors in adults with stroke assessed by calculating p value

Formulas used are:

1. Standard Error (SE)= $\sqrt{\frac{P_1Q_1}{n_1} + \frac{P_2Q_2}{n_2}}$

2. Normal deviate $= \frac{x - \bar{x}}{SE}$

- p1 denotes the %of patients with the specific risk factor
- q1 denotes %of patients without the specific risk factor
- p2 is the %of control population with the specific risk factor
- q2 is the %of control group without risk factor
- n1 is the number of study population
- n2 is the number of control group
- X individual variable
- $\bar{x}$ population mean
- S E. standard error of mean. If Normal deviate > 2, p value should be < 0.05 then that risk factor is statistically significant.[21 8]

3. Standard error of difference between two mean

$$\text{S.E of difference between two mean} = \sqrt{\frac{0_1^{-}}{n_1} + \frac{0_2^{-}}{n_2}}$$

0_1^{-} Standard deviation of study

0_2^{-} Standard deviation of control

n 1 number of study

n2 number of control

The actual difference between two mean is more than twice the standard error of difference between two mean is significant.[21]

REVIEW OF LITERATURE

REVIEW OF LITERATURE

GENERAL CONSIDERATIONS

DEFINITION

According to WHO “stroke is defined as rapidly developing clinical signs of local or global disturbance of cerebral function lasting more than 24hrs. or leading to death with no apparent cause other than vascular”.

The term Transient Ischemic Attack (TIA) implies complete recovery of cerebral function within 24 hrs. Typically neurological signs and symptoms of a transient ischemic attack last for 5 to 15 minute definition must last 24 Hrs. Some of the sub types of stroke include cerebral hemorrhage, cerebral infarction and sub arachnoid hemorrhage.

The normal functioning of the brain is dependant upon a relatively constant supply of oxygen, glucose and other nutrients derived from the blood perfusing it. Normal blood flow is brain to 55 – 70 ml/min. A fall in cerebral blood flow to zero causes deaths brain tissue with 4 to 10 minute. Value <16 to 18ml/100g tissue for cause infarction within an Hour and value < 20ml/100g tissue per minute

cause ischemia without infarction unless prolonged for several Hours or days.

The mean arterial blood pressure, cerebrovascular and tissue resistance local metabolic products (pH, PaO^2, etc) together with several known and unknown factors help to maintain the critical threshold of blood flow for energy metabolism. Further more the blood flow varies in different areas of the brain and auto regulation determines the regional blood flow to meet local metabolic need.

BLOOD SUPPLY TO THE BRAIN

At rest the brain which is only 2 percent of total body weight receives 15 percent of the cardiac output and consumes about 25 percent of the total inspired oxygen. This rich blood supply is carried by two internal carotid and two vertebral arteries which anastamose at the base of brain to form the ‘circle of Willis’. The carotid arteries supply the anterior and the vertebro basilar arterial system supplies the posterior portion of the brain.

The branches of the internal carotid artery are

a. The Ophthalmic artery
b. Anterior cerebral artery
c. Middle cerebral artery
d. Anterior choroidal artery
e. Posterior communicating artery

The vertebral artery which arises from the subclavian artery enters foramen magnum and unites with the opposite vertebral artery at the pontomedullary junction to form the basilar artery. Vertebral artery gives rise to anterior and posterior spinal arteries, the posterior inferior cerebellar artery and small penetrating arteries to the medulla. The basilar artery ascends up to the pontomidbrain junction in the interpeduncular cistern and divides into the two posterior cerebral arteries. Numerous small branches penetrate the brainstem and cerebellum. It also gives rise to the anterior inferior cerebellar artery, the internal auditory artery and the superior cerebellar artery.

The meninges are supplied by branches of the internal carotid, external carotid and vertebral arteries.

THE COLLATERAL BLOOD SUPPLY TO THE BRAIN

Normally each ICA provides blood to the anterior two thirds of the cerebral hemisphere on the ipsilateral side. There is little mixing of blood via the posterior communicating artery with the vertebrobasilar system. However collateral circulation can develop distal to major artery occlusion. The development of collaterals is more effective if the vessel occlusion occurs insidiously rather than suddenly. But unlike the normal cerebral blood supply the functional capacity of the collateral blood supply to respond to changes in perfusion pressure is limited.

Collateral blood flow may develop via

1. The circle of Willis. However about 50 percent of circles have one or more hypo plastic segments (usually one of the communicating arteries) and also since atheroma commonly affects the circle of Willis, the potential for collateral flow is not always good.
2. Around the orbit, branches of the ECA anastomose with branches of the ophthalmic artery if the ICA is severely stenosed.

3. Muscular branches of the vertebral artery in the neck distal to an Obstruction of that artery, receive blood from occipital and ascending pharyngeal of ECA.
4. Leptomeningeal anastomoses on the surface of the brain may develop between cortical branches of the anterior, middle and posterior cerebral arteries.
5. Dural anastomoses can develop between meningeal branches of the precapillary bed of the vertebral arteries.
6. Parenchymal anastomoses occasionally develop in the precapillary bed of the perforating arteries supplying the basal ganglia (Moyamoya syndrome).

Venous drainage

Venous blood flow peripherally via the superficial cerebral veins and centrally via the deep cerebral veins into the venous sinuses which in turn drain into the internal jugular vein. The cerebral veins are thin walled have no valves and the blood flow are often in the same direction as in the neighboring arteries. There are numerous venous connections between the veins and dural sinuses as well as with the venous system of the meninges, skull, scalp and nasal sinuses so facilitating the propagation of thrombus or spread of infection between these vessels.

EPIDEMIOLOGY

This lagged behind coronary heart disease because

1. Stroke is a disorder of late middle age and elderly where other diseases coexist.
2. Stroke pathologically is more diverse and may be due to intracerebral small vessel disease or embolism from the heart or primary intracerebral hemorrhage.
3. Lack of CT was one more reason.

Some of the important epidemiological studies are as follows

One of the classical studies reported from India is the study Conducted at CMC, Vellore by Abraham et al (1970).

According to this study, the incidence of hemiplegia is 68.5 in males and 44.8 in females per 1, 00,000.

Agarwal et al (1976) noted arcus senilis, obesity, TIA, impaired cardiac function and positive family history of cerebrovascular disorder as additional risk factors.

The other prevalence figures available are as follows.

(From epidemiology of stroke in India – Indian college of physician 1999)

City/Area	Rural/ Urban	Sample size	Number of Hemiplegia	Crude prevalence Rate per 1,00,000	Age adjusted
Kashmir	U	63,645	91	143	274
Rohtak	U	79,046	35	44	46
Bombay (Parsi)	U	14,010	118	843	424
Bombay (General)	U	5,723	14	245	NA
Vellore	Semi urban	2,58,576	147	57	84

Regarding extra cranial vascular disease, very few reports are available from India. Nagaraja and Manohar (1985) evaluated 100 patients with cardiac insufficiency by using Doppler Ultrasonography. They noted a maximum incidence of ischemic strokes in the 4^{th} and 5^{th} decades.

MORTALITY

Mortality rises rapidly with age.

INCIDENCE

Stroke incidence rises with age.

Cerebral infarctions account for the vast majority of stroke than subarachnoid and intracerebral hemorrhages.

Geographical racial and social influences

Primary intra cerebral hemorrhage is less common in Western countries than in Japan and China.

Trends in mortality and incidence

Incidence is declining with assumption that the prevalence of hypertension is less than it was and early diagnosis and treatment of hypertension may also be responsible. The prevalence of Rheumatic heart disease also is less than it was and earlier.

Seasonal and diurnal variation

Stroke incidence and mortality is more in winter, the possible explanation being the effect of temperature, pollution and higher blood pressure in the winter. The increased mortality may also be

due to complications of stroke such as pneumonia which is more common in winter.

Cerebral infarction occurs most commonly in the hour or two after waking in the morning. Subarachnoid hemorrhage is unlikely to occur during sleep. Circadian changes in physical activity, catecholamine level, blood pressure, blood viscosity, platelet aggregation may explain. Intracerebral hemorrhage is more likely to occur during strenuous activity than during rest or sleep.

ETILOGY OF STROKE

Common causes

Thrombosis

Lacunar stroke , Large vessel thrombosis

Embolic occlusion

Artery to artery

Carotid bifurcation, Aortic arch , Arterial dissection

Cardio embolic

Atrial fibrillation

Mural thrombosis

Myocardial infarction

Dilated cardiomyopathy

Valvular lesion

Mitral stenosis

Mechanical valve

Bacterial endo carditis

Paradoxical embolus

Atrial septal defect

Patent foramen ovale

Atrial septal aneurysm

Cardio embolic stroke

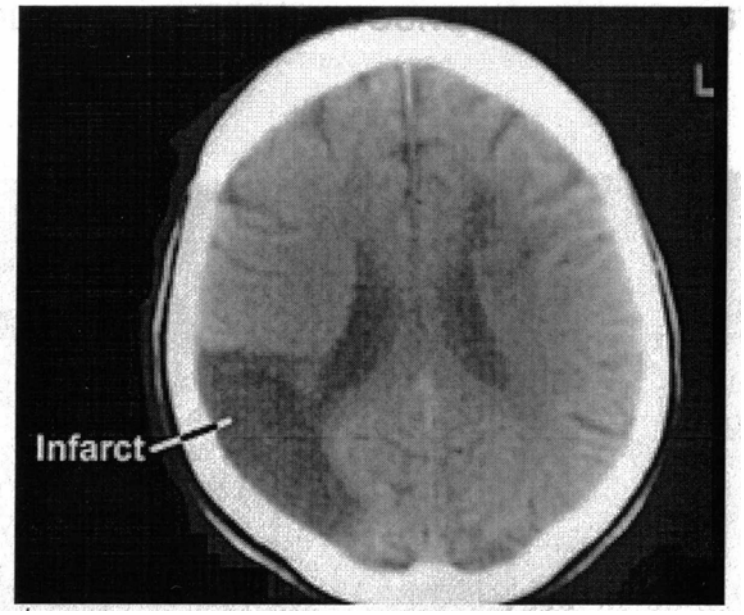
Stroke caused by heart disease is due to embolism of thrombotic material forming on the arterial or ventricular wall or left heart valve .the thrombus may fragment or lyse quickly, producing only stroke. Occlusion may longer, producing stroke.

Emboli from heart most often lodge in the Middle cerebral artery, the posterior cerebral artery, or one of the branches; infrequently, the anterior cerebral artery territory.

Artery to artery embolic stroke

Thrombus formation on atherosclerosis plaques may embolize to intra cranial arteries producing an Artery to artery embolic stroke. A diseased vessels may acutely thrombose; the most common

CT SCAN BRAIN - INFARCT



source of embolism is the carotid bifurcation , aortic archand common carotid artery ,vertebral artery , and basilar artery.

Carotid atherosclerosis

Atherosclerosis within the carotid artery occurs most frequently within common a carotid artery bifurcation and proximal internal carotid artery . Male gender, older age , smoking , older age , hypertension , diabetes , and hypercholesterolemia are risk for carotid disease. As they are for stroke general

Cerebral Venous thrombosis

Occlusion of intracranial venous sinuses or cortical veins can cause infraction or hemorrhage. Spread of infections of the ear and Para nasal sinuses produces septic thrombosis. Aseptic thrombosis of the cerebral veins has been associated with –

1. Pregnancy, peurperium and use of oral contraceptives.
2. Cardiac disease with congestive cardiac failure.
3. Diabetes mellitus.
4. Dehydration.
5. Hematological diseases.

Stroke during pregnancy and puerperium

This is one of the most common causes of stroke in young women. About 65% of nonhaemorrhagic hemiplegics of pregnancy are due to arterial rather than venous occlusion. Alterations in clotting factors during pregnancy and in the post-partum period (a decrease in fibrinolysis and an increase in fibrinogen) lead to a hypercoagulable state. Other causes of stroke during pregnancy and peurperium are –

1) Venous sinus and cortical vein thrombosis.
2) Emboli from mural thrombi of peripartum cardiomyopathy.
3) Intracerebral and subarachnoid hemorrhage due to eclampsia and consumptive coagulopathies of peripartum period which may occur due to amniotic fluid embolism, premature separation of placenta, septic abortion, hydatidiform mole, intrauterine fetal death and uterine rupture.

Hematological Disorders

The disorders associated with stroke include –

1) Hyper viscosity syndromes.
2) Sickle cell anemia.
3) Polycythaemia.

4) Paroxysmal nocturnal haemoglobinuria.

5) Disorders of platelets of blood coagulation.

6) Leukemia.

Hyper viscosity syndrome is most often associated with multiple myeloma with an increase in lgG or lgA Para proteins which causes hyperviscosity, small vessel occlusion and multiple areas infarction or hemorrhage.

Sickle cell disease can cause ischemic infarction, intracerebral hemorrhage, venous sinus and cortical vein thrombosis and subarachnoid hemorrhage. The overall incidence of stroke in sickle cell disease is between 6% to 15%. The risk of cerebral infraction is greatest in children with sickle cell anemia. Stroke occurs infrequently in sickle cell patients older than 20 years complications are more common in adults. In addition to small vessel occlusion from intravascular sickling, endothelial proliferation affects small arteries and arterioles and angiopathy may involve the anterior part of the circle of Willis. Stroke recurrence is common in sickle cell anemia and may be avoided by periodic exchange transfusions aimed at keeping hemoglobin-S levels below 20%.[2]

In polycythaemia, there is a predispositions both arterial and venous thrombosis and retinal vein occlusion. Intracerebral and subarachnoid hemorrhages may occur occasionally. Paroxysmal nocturnal haemoglobinuria may result in cerebral venous thrombosis and is suspected in patients with chronic hemolytic anemia, unexplained pain and multiple episodes of venous thrombosis at different systemic sites.

Amount the platelet and coagulation disorders resulting in stroke are chronic idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura, idiopathic thrombocytosis and disseminated intravascular coagulation. Chronic idiopathic thrombocytopenic purpura is three to four times more common in women than in men and may occasionally result in intracerebral hemorrhage.

The triad of thrombotic thrombocytopenic purpura includes thrombocytopenic purpura, haemolytic anemia and focal cerebral signs. Seizures are also common. The disorder may start in the second half of pregnancy simulating eclampsia. The small cerebral vessels demonstrate hyperplasia and platelet thrombi.

In idiopathic thrombocytosis recurrent cerebralheamorrhage, thrombosis or both are associated with an elevated platelet count, megakaryocytic hyperplasia of the bone marrow and some-times splenomegaly.

Disseminated intravascular coagulation may result in either cerebral hemorrhage or thrombosis. It involves the consumption of coagulation factors and platelet and may be associated with carcinoma, disorders of peripartum period and sepsis.

Chronic inflammatory bowel diseases such as ulcerative colitis and regional enteritis have been associated with hypercoagulable state and thrombocytosis predisposing to recurrent retinal artery branch occlusion, cerebral venous and arterial thrombosis.

Isolated reports of occlusive cerebrovascular diseases have appeared in association with elevation of plasma factor VIII and deficiency of factor XII. Platelet hyperaggregability and occlusive cerebral vascular disease may occur with the use of estrogens or in patients with migraine

VASCULAR DISEASES

Apart from atherosclerosis there are other vascular diseases which can produces stroke They are –

1) Arteritis due to –

a) Collagen disorders.

b) Infection- Meningovascular syphilis

Tuberculous arteritis

Pyogenic infection

Fungal- mucormycosis

c) Takayasu's arteritis

d) Drug included vasculitis-methamphetamine

e) Irradiation

2) Ruptured saccuiar aneurysm

3) Ruptured arterio- venous malformations

4) Moyamoya disease

5) Dissection of intracranial cerebral arteries

6) Fibro muscular dysplasia

The collagen vascular disease most frequently causing stroke is systemic lupus erythematosus. There is an increased risk of thrombosis in patients with an immunoglobulin called "lupus

anticoagulant.” The incidence of stroke is less in polyarteritis nodosa with 13% reportedly experiencing cerebal infarction or hemorrhage. Stroke is infrequent in scleroderma and rheumatoid arthritis.

Meningeal infections can result in cerebral infarction through development of inflammatory changes in the vessel walls, Meningovascular syphilis and tuberculous infections are common causes. Pyogenic infections are infrequent causes. Another infection that may result in cerebral arteritis is mucor-mycosis. The fungus obtains access to the brain through with blood dyscrasiasis and in immunosuppressed patients. Other rate causes of cerebral infarction are typhus, schistosomiasis, falciparum malaria and trichinosis.

Takayasu arteritis (pulses less disease or aortic arch syndrome) is a giant cell arteritis that may result in narrowing and thrombosis of large branches of the aortic arch at their origin and aneurismal formation. Sings of ischemia of the head and arms include cataracts, retinal and optic atrophy, transient monocular blindness, focal cerebral symptoms, hypertension in the legs with intermittent claudication in the arms (reverse coarctation).

Drugs that have been associated with stroke include methamphetamines, LSD, heroin, oral contraceptives, and anticoagulants; Methamphetamine induces a necrotizing vasculitis that may result in intracerebral, subdural or subarachnoid hemorrhages. LSD which is an ergot alkaloid produces arteriospasm and heroin produces allergic vascular hypersensitivity leading to cerebral infarction. The risk of stroke with the use of oral contraceptives is increased five to nine fold for thrombosis and two fold for hemorrhage.

Saccular aneurysms are small thin-walled blisters protruding from the arteries of circle of Willis or the major branches arising from there. Owing to the local weakness, the intima bulges outward, the sac gradually enlarges until finally dissolution of the wall and rupture occur.

Moyamoya disease is a syndrome of stenosis of the vessels in and around the circle of Willis, with profusion of telangiectatic collateral vessels at the base of brain that have angiographic appearance of a puff of smoke, Microaneurysms may develop and either infarction or subarachonoid haemorrhage may result.

Dissection of intracranial cerebral arteries most often affect the middle cerebral and basilar arteries with the clinical presentation of acute infarction. These subintimal dissections of unknown cause develop in otherwise healthy individuals. Dissection of the major extra cranial cerebral, arteries may be spontaneous dissections occur most often in patients younger than 40 years.

Fibro muscular dysplasia is a non-atheromatous vascular disorder in which there is intimal and medial fibroplasias of the extra cranial internal carotid artery and other large systemic vessels. The intracranial arteries are usually spared. The disorder is far more common in women. The cause is not understood.

Homocystinuria

Homocystinuria predisposes to cerebral arterial or less often venous thrombosis as well as to thromboemboli in other organs. The mechanisms are not delineated but the estimated risk of stroke at a young age is between 10% and 16%. The diagnosis is usually made in childhood because of other stigmata such as thin Marfan-like appearance, malar flush, dislocated ocular lens, bony deformities, mental retardation and seizures.

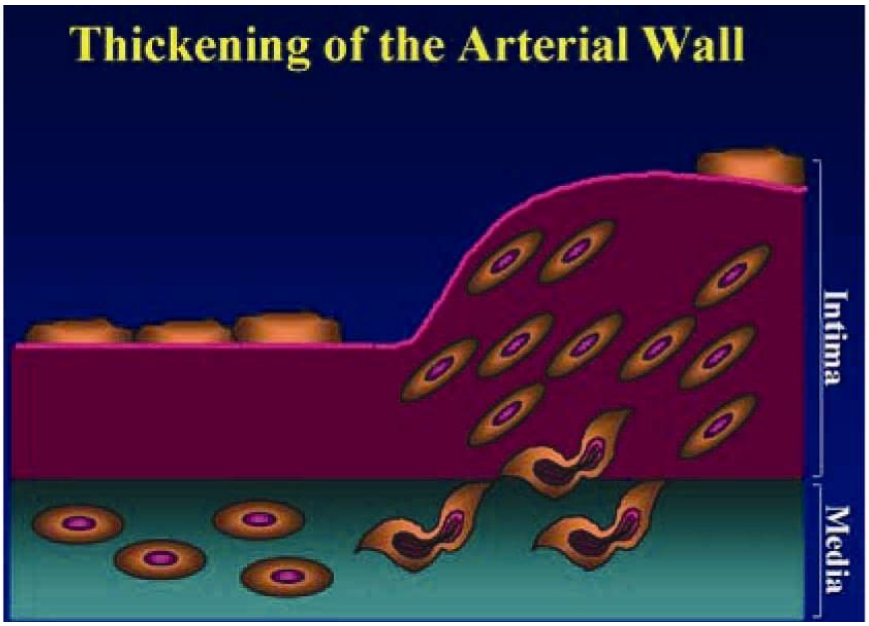
ATHEROSCLEROSIS

It is leading cause of death and disability in the developed world. It commonly causes myocardial infarction and angina pectoris. Atherosclerosis of the arteries supplying the central nervous system frequently provokes strokes and transient cerebral ischemia. In the peripheral circulation, atherosclerosis causes intermittent claudication and gangrene and jeopardizes limb viability. Even within a given arterial bed, Atherosclerosis tends to occur focally. In the coronary circulation for example the Proximal left anterior descending coronary artery exhibit a particular predilection for developing atherosclerotic occlusive disease. Atherosclerosis affects the proximal portion of the renal arteries and in extra cranial circulation to the brain, the carotid bifurcation. The clinical expression of atherosclerosis may be **chronic**, as in the development of stable, effort –induced angina pectoris. **Acute** clinical event such as Myocardial infarction, a cerebrovascular accident or sudden cardiac death.

Fatty streak formation

Fatty streak represent initial lesion of atherosclerosis. The formation of these early lesions of atherosclerosis most often arise

ATHERSCLEROSIS



from focal increases in the content of lipoprotein within regions of the intima. This lipoprotein may collect in the intima of arteries.[4]

Lipoprotein oxidation

Lipoprotein sequestered from plasma antioxidant in the extra cellular space of the intima because susceptible to oxidative modification. Modification of lipid may include formation of hydro peroxides , lysophospholipids, oxysterol and aldehyde breakdown products of fatty acids. A more recently recognized modification may result from local hypocholorous acid production by inflammatory cells within plaque.[4]

Non enzymatic glycation

In diabetic patients with sustained hyperglycemia, non enzymatic glycation of apolipoprotein and other arterial protein likely occur that propensity to accelerate atherogenesis.

Leukocyte recruitment

Leukocyte recruitment occurs as a second step in the formation of fatty streak .the white blood cell types typically found in

the evolving atheroma include monocyte andlymphocyte.Constituents of oxidatively modified LDL can augment expression of leukocyte adhesion molecules once adherent to the surface of the arterial endothelial cell via interaction with adhesion receptors, the monocytes and lymphocytes penetrate the endothelial layer and take up residence in the intima in addition to products of modified lipoproteins , cytokines (a class of protein mediators of inflammation) can regulate the expression of adhesion molecules involved in leukocyte recruitment.[4]

FOAM CELL FORMATION

Once resident within the intima , the mononuclear phagocytes differentiate into macrophages and transform into lipid-laden foam cells . The conversion of mononuclear phagocytes into foam cells requires the up take of lipoprotein particles by receptor mediated endocytosis. Lipid accumulation and hence propensity to form atheroma. Macrophages play a vital role in the lipid accumulation in the arterial wall during atherogenesis. Some foam cell may die result of programmed cell death known as **apoptosis.**

This death of mononuclear phagocytes result in formation of lipid rich center, often called **necrotic core.**[4]

RISK FACTOR

Lipoprotein disorder as predisposing factor for atheroma formation, other etiologies may modulate atherogenesis.Hypertension constitutes an independent risk factor for coronary event .Male gender postmenopausal state also augment the risk of developing coronary artery disease. Diabetes mellitus aggravates atherogenesis. Diabetes associated dyslipidemias strongly promote atherogenesis. In particular, the constellation of insulin resistance, high triglycerides and low H D L, often in association with central adiposity and hypertension frequently seen in type 2 Diabetes patients, seems to accelerate atherogenesis potently. L p (a) provides a potential link between hemostasis and blood lipid. It consists of an apoprotein (a). Apoprotein (a) has homology with plasminogen and may inhibit fibrinolysis by competing with plasminogen. Another non lipid risk factor for coronary event, elevated level of homocysteine, may act by promoting thrombosis. The relationship between tobacco use and atherosclerosis also remain poorly understood. In human an atherogeneic role for viral or microbial pathogenesis remain speculative.[4]

CAROTID INTIMAL MEDIA THICKNESS

.Most atheroma produces no symptom and many never cause clinical manifestation. During the initial phases of atheroma development, plague usually grows outward in a abluminal direction. Vessels affected by atherogensis tend increase in diameter a phenomenon known as **compensatory enlargement**. a type of vascular modelling.[4] Carotid intima media thickness is one of the atherosclerosis changes.

STROKE SYNDROMES

Stroke Syndrome are defined –

1) By the time course of the disease.
2) By the vascular supply to the area of ischemic brain.

Temporal profile of stroke

Depending upon the time course of the disease the strokes are classified as transient ischemic attacks, reversible ischemic neurological deficit, progressing stroke or stroke in evolution and completed stroke.

Transient ischemic attack (TIA)

It is a sudden focal neurological dysfunction due to ischemia of a portion of the brain and which resolves completely within 24 hours. Most of the episodes last usually less than 10 minutes but attacks lasting many hours are often documentedly associated with embolism. They may herald the oncoming vascular catastrophe. Previous history of TIAs are much more common but very rare in intracerebral hemorrhage. Recurrent attacks of the same pattern indicates that thrombosis is the possible cause while multiple episodes of different pattern indicates embolism.

Symptoms of TIA vary depending upon the vascular territory involved. Symptoms due to involvement of carotid system are motor defects (weakness, paralysis or clumsiness) of one extremity or of both extremities of one side, sensory deficit (numbers or paraesthesia) of one or both extremities of one side, aphasia, amaurosis fugax and homonymous hemianopia. Symptoms due to involvement of vertebrobasilar system are motor defect of any combination of extremities up to quadriplegia or changing from one side to another in different attacks, sensory defects which are bilateral or changing from side to side in different attacks, bilateral loss of vision or homonymous hemianopia and ataxia with or without vertigo.

Reversible ischemic neurological deficit (RIND)

It is a focal ischemic event lasting longer than 24 hour but complete resolution of the deficit within three weeks. These episodes are also referred to as stroke with full recovery.

Progressing stroke or stroke in evolution

The temporal pattern of stroke is sometimes extended. Disability may increase by stepwise progression. Sudden deteriorations are interspersed with static intervals. Less commonly there is slow uninterrupted progression. The full extend of the patient's stroke conveys somewhat greater diagnostic uncertainty. A rapidly growing neoplasm or a subdural haematoma is more often a real differential diagnostic syndrome.

Completer stroke

It is the stable focal ischemic neurological deficit from which recovery occurs gradually over weeks and months.

Specific Vascular Syndromes

Stroke syndromes are defined not only by their temporal profile but also by the vascular supply to the area of ischemic brain.

INTERNAL CAROTID SYNDROME

The cervical portion of the carotid artery is a common site for both severe atheroma and thrombotic occlusion. About 30 percent of all occlusive lesions may be silent or asymptomatic. Symptoms include brief episode of confusion, speech difficulties, sensory paraesthesia with or without motor weakness on the opposite side. Ipsilateral amaurosis fugax, fleeting or semi permanent, alternating with or accompanied by a contra lateral hemiplegia or sensory deficit is pathognomonic of carotid artery syndrome, but it is noted in only 15 to 20 percent of the subjects.

The clinical manifestation may be similar to middle cerebral syndrome. Feeble carotid superficial temporal artery pulsation, dilated pupil and poorly pulsating retinal vessels With or without optic atrophy) on the side of suspected carotid lesion and ocular or cervical bruits on the ipsilateral side may suggest the correct diagnosis.

In the subjects with an old or silent occlusive carotid axis lesion on one side a new lesion on the other side may prove catastrophic. Here physical findings of bilateral hemiplegia (quadriplegia) with coma can be mistaken for basilar artery syndrome.[3]

MIDDLE CEREBRAL SYNDROME

The cortical branches supply most of the lateral surface of the cerebral hemisphere, except for the regions supplied by the anterior and posterior cerebral arteries. The areas of irrigation include the sensory-motor cortex, the motor and sensory speech centers, auditory area and visual radiation. The penetrating branches (lenticulostriate arteries) supply the putamen, globuspallidus, genu and posterior limb of the internal capsule.

The clinical picture of middle cerebral artery occlusion is variable. Contra lateral hemiplegia, hemianaesthesia with or without homonymous hemianopia and aphasia (dominant and non dominant) is the common outcome. However, occlusive of the upper division results in contra lateral hemi paresis with sensory deficit and expressive aphasia (Broca's aphasia) whereas dominant side. Even monoplegic symptoms can occur with an occlusive lesion of a single cortical branch.

Occlusive of penetrating branches (lenticulostraite arteries) has been blamed for a dense sensory motor hemiplegic syndrome (capsular hemiplegia) but significant sensory loss seldom occurs with

such an occlusion, whereas pure motor hemiplegia is not uncommon.[3]

ANTERIOR CHOROIDAL SYNDROME

This artery supplies the posterior limb of the internal capsule, which carries the corticospinal and sensory fibres for the contra lateral limb. This syndrome which represents a true capsular hemiplegia (dense hemiplegia, hemianaesthesia and homonymous hemianopia) is rare.

Anterior cerebral syndrome

These cortical branches mainly supply the medial superior surface of the frontal lobe and the parietal lobe up to the Para central lobule. The penetrating branched supply the anterior limb of the internal capsule and part of the head of the caudate nucleus.

Anterior cerebral artery occlusion proximal to the anterior communicating artery, in subjects with a symmetrical circle of Willis, is frequently asymptomatic. Occlusion distal to the anterior communicating artery manifests itself by sensory motor paralysis of the opposite lower extremity with mild weakness of the opposite shoulder. Mental changes, rectal and urinary incontinence, gait

disturbances, apraxia and grasp and sucking reflexes may accompany the above findings.

Occlusion of an unpaired anterior artery (supplying both the hemispheres) results in a cortical type of paraplegia, with sphincter incontinence and a mental state in which the patient is alert but mute (akinetic mutism). Aphasia and hemianopia are never seen.

Occlusion of the penetrating branches and of the Heubner's artery is frequently blamed for ataxic tremor of the contra lateral limbs (frontal ataxia) Apraxia/ideomotor dyspraxia of the limbs and gait may also be present.[3]

POSTERIOR CEREBRAL SYNDROME

This artery supplies the medial and inferior aspects of the occipital and temporal lobes. Its branches also supply the mid-brain, cerebral peduncle and most of the thalamic and subthalamis regions.

Thrombotic occlusion of the posterior cerebral arteries is relatively rare. Contra lateral homonymous hemianopia is a significant finding and this results from infarction of the primary visual area (calcarine, cortex) the central vision is frequently spared, even in

patients with bilateral disease (gun barrel vision). Other manifestations of visual dysfunction include illusory or distorted vision visual object agnosia and various forms of dyslexia without dysgraphia. The pupillary reflexes are well preserved contra lateral hemiplegia from a lesion of the cerebral peduncle (pedunclar hemiplegia) and thalamic syndrome, there is varying degree of sensory loss to all modalities and spontaneous burning or agonizing pains are frequent (analgia dolorosa) Memory loss (amnesia) denotes a lesion of the medial temporal cortex. Contra lateral involuntary choreoathetosis or ataxic tremors are rarely observed.

Vertebro-basilar syndrome

After traversing through the bony vertebral canals, both vertebral arteries unite intracranially to form the basilar trunk. Their short paramedian and long circumferential branches supply the entire brainstem, cerebellum and the vestibular apparatus. Ischemic disorders, therefore manifest by episodes of vertigo, dizziness diplopia, dysarthria, dysphasia, incoordination of gait and limbs and bilateral signs of sensory motor deficit. Occipital headaches may be present.

Ipsilateral IIIrd nerve palsy (dilated pupil, ptosis and external strabismus) with contra lateral hemiplegia (Weber's Syndrome) or with crossed cerebeller ataxia (Claude's syndrome) is diagnostic of mid-brain localization. Homolateral paralysis of the Vith and VIIth nerves (internal squint and facial palsy) with contra lateral hemiplegia and hemianaesthesia (Millard-Gubler syndrome) is suggestive of a pontine lestion. Palatal paralysis and ataxia of limbs with impairment of posterior column sensation on one side of the body together with diminution of pain thermal sense on the opposite limbs (Wallenberg's syndrome) indicate lateral medullary infarction.

Not infrequently quadriplegia with bilateral conjugate, lateral gaze palsy and mute state but with fully preserved consciousness has been described (locked in syndrome) and suggests infarction of the basis pontis (separating the tegmentum) from midbasilar occlusion.

Occlusion of isolated cerebellar branches may product dizziness, nausea, vomiting, nystagmus and appendicular or truncal ataxia without sensory-motor deficit in any limb. Such a syndrome should be differentiated from cerebeller hemorrhage where emergency surgical decompression proves lifesaving.[3]

Lacunar Syndromes

Occurs as a result of infarcts in the deep portions of cerebral hemispheres and brain stem due to occlusion of small perforating branches. Pure motor hemiplegia is the most common lacunar syndrome due to an infarct in the posterior limb of internal capsule. There is no sensory deficit, visual field defect or aphasia.

Arteritis

The clinical features of syphilitic and other forms of arteritis involving various cerebral arteries are in to way different from the neurovascular syndrome described under cerebral thrombosis. With specific serological tests, the diagnosis of meningovascular syphilis is not difficult.

CLINICAL PRESENTATION

In stroke patients, symptoms and signs are in relation to the arterial circulation involved. The clinical assessment may be corroborated by diagnostic studies such as computed tomography scanning or carotid angiography. However use of these and other diagnostic techniques may be, the stroke syndrome continues to be characterized by the nature and time course of the patient's clinical

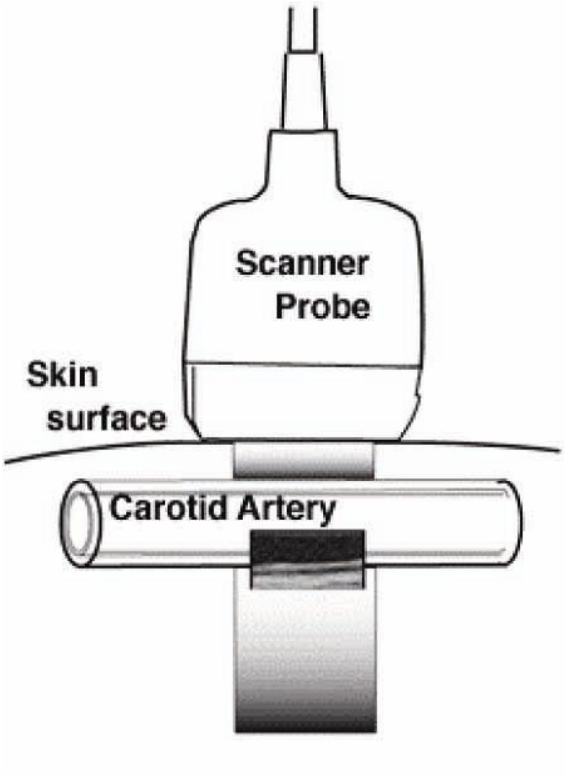
findings. A careful analysis of history may help in differentiating stroke due to thrombosis, embolism or hemorrhage.

Onset and Progression of stroke

Patient's activity during the onset of stroke indicates the possible cause of stroke. Intracerebral hemorrhage usually presents abruptly when the patient is awake and is prone to occur while he or she is engaged in physical exertion. Severe headache may precede the onset of hemorrhagic stroke by several hours and loss of consciousness is a usual feature.

In cerebral embolism the full blown picture of stroke evolves within a few seconds "like a bolt out of the blue" without any warning symptoms. In cerebral thrombosis the focal disability may occur at any time. The patient may awake with an established lesion very frequently. The deficit evolves during a period of 1 or 2 days. Loss of consciousness occurs very rarely though drowsiness is common. Severe headache is unusual. Sometimes cerebral infarctions due to thrombosis and those due to embolism may be indistinguishable in terms of the time, course and nature of neurological findings.

ULTRA SONO GRAPHY CAROTID INTIMAL MEDIA THICKNESS MEASUREMENT



Fits

Epileptic fits, generalized or of the focal type may occur at the beginning or during extension of a stroke. Fits may occur in thrombosis, embolism or hemorrhage and it is not helpful in differentiating these conditions. Fits commonly occur in cerebral venous thrombosis.

Signs of meningeal irritation

Neck stiffness occurs when blood enter into CSF in case of intracerebral hemorrhage or when there is rupture of saccular aneurysm producing subarachnoid hemorrhage,. In our country since tuberculous meningitis is a common disease, it should be considered in stroke patients with signs of meningitis.

Arterial Pulsations and bruit

A significant reduction in cerebral blood flow can occur due to atherosclerotic lesion narrowing the internal carotid artery or vertebral artery lumen by more than 60 to 80% of cross sectional area. Severe lesion of this kind are frequently seen in the internal carotid artery near the carotid sinus and in the vertebral channels coursing over the forehead and supra-orbital and supratrochlear arterial pulsations on

the rim of the orbit suggest carotid occlusion. An additional sign of carotid occlusion is the presence of ocular bruit – an intracranial murmur over the opposite carotid artery heard over the eye ball. Though vertebral artery is not accessible for palpation, its occlusion at its origin is suggested by a bruit heard over supraclavicular fossa.

Cardiogenic embolic occlusion

The determination that the stroke is embolic in origin generally results from the demonstration of an appropriate cardiac abnormality in a patient with a stroke featuring characteristics suggestive of an embolus. Clinical features of a stroke which suggest a possible cardiac embolic origin are –

1) Sudden onset with maximal neurological deficit appearing immediately.
2) History of multiple episodes of TIA of different pattern.

Multifocal cerebral infarctions especially in a patient with an associated systemic arterial occlusion, (e.g.) renal arteries or limb arteries.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

Table – 1

SEX INCIDENCE OF ADULTS WITH STROKE IN 50 PATIENTS

Sex	Number of cases	Percentage
Male	32	64%
Female	18	36%
Total	**50**	**100%**

Table – 2

AGE INCIDENCE OF STROKE IN 50 CASES

Age	20-30	31-40	41-50	51-60	> 60
No. of Cases	2	6	12	16	14
Percentage	4	12	24	32	28

AGE INCIDENCE OF STROKE IN 50 CASES

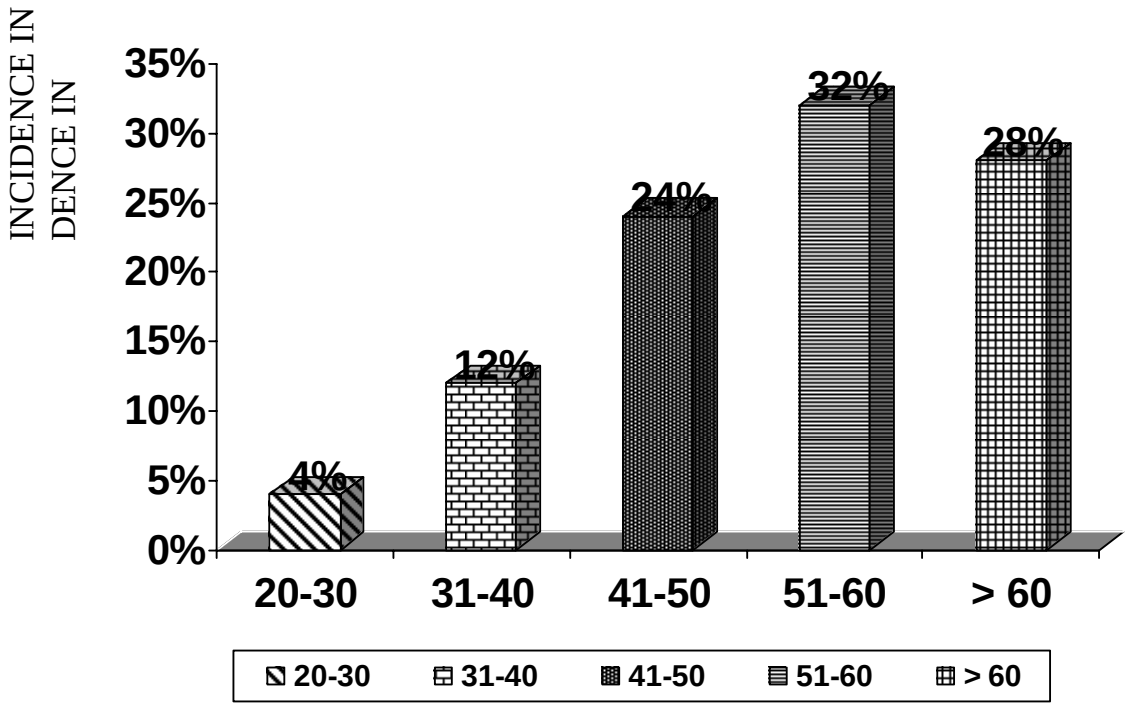


Table – 3

PREVALENCE OF RISK FACTORS IN 50 ADULTS WITH STROKE

Risk Factors	Number of cases	Percentage
Systemic Hypertension	16	32%
Smoking	10	20%`
Diabetes mellitus	6	12%
Tuberculous Meningitis	5	10%
Atrial Fibrillation	4	8 %
Hyper Lipidemia	4	8%
Uncertain Cause	5	10%

Risk factors were present in 90% of the cases

20% had more than one risk factor

20% of males with stroke were smokers.

PREVALENCE OF RISK FACTORS IN 50 ADULTS WITH STROKE

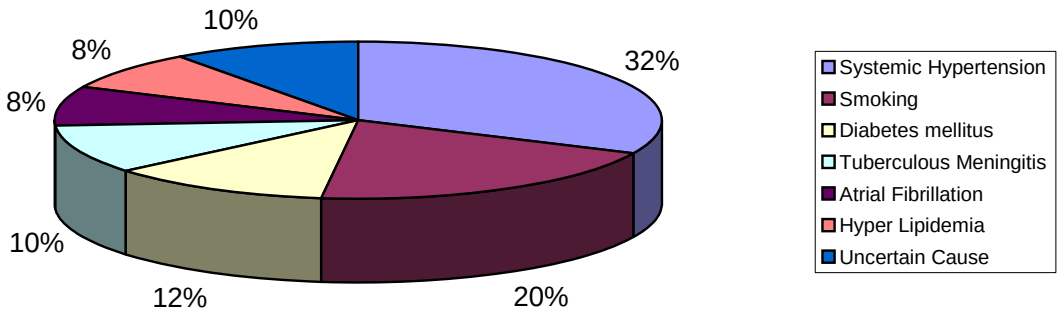


Table – 4

SITE OF LESION IN 50 CASES (CLINICAL AND CT SCAN EVALUATION)

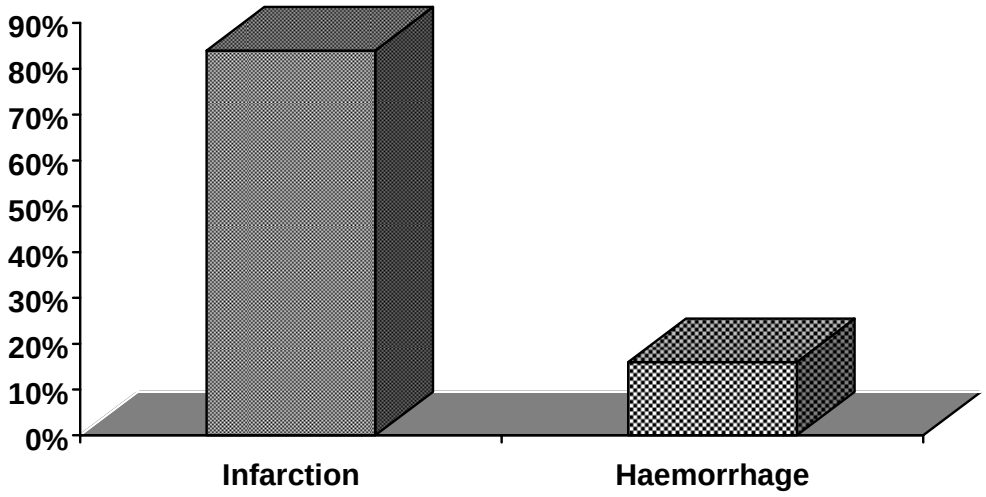
Site of Lesion	Number of cases	Percentage
Middle Cerebral Artery territory	40	80%
Vertebrobasilar Artery territory	5	10%
Anterior Cerebral Artery territory	5	10%

Table – 5

TYPE OF STROKE (CLINICAL AND CT SCAN EVALUATION)

Type of Lesion	Number of cases	Percentage
Infarction	42	84%
Haemorrhage	8	16%

PATHOLOGICAL LESION IN ADULTS WITH STROKE



TYPES OF STROKE

DATA ANALYSIS

DATA ANALYSIS

ANALYSIS OF SIGNIFICANCE OF RISK FACTORS IN ADULTS WITH STROKE DONE BY 'P' VALUE CALCULATION:

1. SIGNIFICANCE OF HYPERTENSION IN ADULTS WITH STROKE

HYPERTENSION	PRESENT	ABSENT
STUDY GROUP $n_1 = 50$	32 %	68%
CONTROL POPULATION $n_2 = 50$	2%	98%

1. Standard Error= $\sqrt{\frac{p_1q_1}{n_1} + \frac{p_2q_2}{n2}}$

2. Normal deviate $= \frac{x - \bar{x}}{S.E}$

Normal deviate 4.34, hence 'p' value should be < 0.05.

So it is statistically significant

2. SIGNIFICANCE OF SMOKING IN ADULTS WITH STROKE

SMOKING	PRESENT	ABSENT
STUDY GROUP $n_1 = 50$	20%	80%
CONTROL POPULATION $n_2 = 50$	4%	96%

Normal deviate 2.42

'p' value < 0.05.

Hence the study value is statistically significant

3. SIGNIFICANCE OF ATRIAL FIBRILLATION IN ADULTS WITH STROKE

ATRIAL FIBRILLATION	PRESENT	ABSENT
STUDY GROUP $n_1 = 50$	8%	92%
CONTROL POPULATION $n_2 = 50$	0%	100%

Normal deviate 2.16

'p' value < 0.05.

Hence the study value is statistically significant

4. SIGNIFICANCE OF HYPERLIPIDEMIA IN ADULTS WITH STROKE

HYPERLIPIDEMIA	PRESENT	ABSENT
STUDY GROUP $n_1 = 50$	8%	92%
CONTROL POPULATION $n_2 = 50$	0%	100%

Normal deviate 2.16

'p' value < 0.05.

Hence the study value is statistically significant

5. SIGNIFICANCE OF TUBERCULOUS MENINGITIS IN ADULTS WITH STROKE

TUBERCULOUS MENINGITIS	PRESENT	ABSENT
STU DY GROUP $N_1 = 50$	10%	90%
CONTROL POPULATION $N_2 = 50$	0%	100%

Normal deviate 2.35

'p' value < 0.05.

Hence the study value is statistically significant

6. SIGNIFICANCE OF DIABETES MELLITUS IN ADULTS WITH STROKE

DIABETES MELLITUS	PRESENT	ABSENT
STUDY GROUP N_1 = 50	12%	88%
CONTROL POPULATION N_2 = 50	0%	100%

Normal deviate 2.55

'p' value < 0.05.

Hence the study value is statistically significant

7. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS Mm	STANDARD DEVIATION
STUDY N1 50	**0.65**	**0.30**
CONTROL N2 50	**0.50**	**0.20**

Standard error difference between two mean is 0.05 which is thrice than difference between carotid intima media thickness in study and control group. So, it is significant.

7. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN HYPERTENSION WITH STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS Mm	STANDARD DEVIATION
STUDY N1 16	0.69	0.33
CONTROL N3 50	0.50	0.20

Standard error difference between two mean is 0.08 which is twice than difference between carotid intima media thickness in study and control group .So it is significant.

8. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN HIGH LDL WITH STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS Mm	STANDARD DEVIATION
STUDY N1 4	0.74	0.35
CONTROL N2 50	0.50	0.20

Standard error difference between two mean is 0.09 which is twice More than differences between mean carotid intima media thickness in study and control group So it is significant.

9. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN HYPER TRIGLYCERIDE WITH STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS Mm	STANDARD DEVIATION
STUDY N1 4	0.74	0.35
CONTROL N2 50	0.50	0.20

Standard error difference between two mean is 0.09 which is twice more than difference between mean carotid intima media thickness in study and control group So it is significant.

10. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN LOW HDL WITH STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS mm	STANDARD DEVIATION
STUDY N1 4	0.74	0.35
CONTROL N2 50	0.50	0.20

Standard error difference between two mean is 0.09 which is twice more than difference between mean carotid intima media thickness in study and control group. So it is significant.

11. SIGNIFICANCE OF CAROTID INTIMA MEDIA THICKNESS IN DIABETES MELLITUS WITH STROKE

	MEAN CAROTID INTIMA MEDIA THICKNESS mm	STANDARD DEVIATION
STUDY N1 6	0.71	0.38
CONTROL N2 50	0.50	0.20

Standard error difference between two mean is 0.08 which is twice more than difference between mean carotid intima media thickness in study and control group. So it is significant.

ANALYSIS OF TREATMENT OUTCOME AND PROGNOSIS

Treatment of stroke includes –

1) Acute therapies designed to minimize brain infarction and life supportive measures.

2) Rehabilitative aimed at improving the quality of life.

3) Treatment aimed at preventing recurrence of strokes.

None of the patients included in this study had complete loss of consciousness though drowsiness was present in ten patients. All the fifty patients presented with completed stroke with fixed neurological deficits, admitted several hours to a few days after the onset of stroke. Since there were no patients with stroke in evolution, anticoagulant therapy was not used to minimise brain infarction. Measures to reduce cerebral oedema with intravenous mannitol, frusemide and steroids were undertaken in fifty patients.

Anticoagulant therapy with intravenous heparin was started in four patients who had rheumatic mitral valve disease with atrial fibrillation.

Five patients with tuberculous meningitis with stroke were treated with rifampicin, INH, ethambutol and pyrazinamide along with steroids. Blood pressure of the sixteen hypertensive patients was controlled by oral administration of enalapril.

Antiplatelet therapy with low dose aspirin was given in all patients who presented with thrombotic stroke.

Physical rehabilitative measures to maximise functional recovery were undertaken in all patients.

Prevention of recurrence of stroke was by long term antiplatelet therapy in those with thrombotic stroke, oral anticoagulant, therapy in those with aseptic cardiogenic emboli and control of blood pressure in hypertensive. Prophylactic antibiotics before any minor or major surgical procedures in susceptible cardiac patients minimises the risk of sub acute bacterial endocarditis.

Of the Fifty patients, fourty showed stable unchanged course over the first 7 days. Ten patients showed improvement in the first 7 days. None of the patients showed worsening after admission and no patient died.

DISCUSSION

DISCUSSION

In our study 50 adults with stroke were included.

Incidence of stroke was more common in males than females. The incidence in males was 64% when compared to 36% in females.

The peak incidence of stroke was observed in the age group 51-60 years which was 32%. Age group > 60 years is also 28%.

In our study 84% of stroke was above 40 years of age.

ANALYSIS OF RISK FACTORS

Hypertension

Hypertension is the most common risk factor associated with stroke. The incidence of hypertension was 32% in our study. In a study conducted by P.A.G. SANDER COCK et al, the incidence was 32%[11]. It correlates with our study.

Diabetes mellitus

It is one of the risk factor in stroke. Out of 50 patients studied 6 patient had diabetes mellitus.

Case-control studies of stroke patients and prospective epidemiological studies have confirmed an independent effect of diabetes with a relative risk of ischemic stroke in persons with diabetes from 1.8 to 3.0. Among Hawaiian Japanese men in the Honolulu Heart Program, those with diabetes had twice the risk of thromboembolic stroke of persons without diabetes that was independent of other risk factors. In a population-based cohort in Rancho Bernardo, persons with diabetes had a risk-factor adjusted relative risk of stroke of 1.8 in men and 2.2 in women. In Framingham, persons with glucose intolerance have double the risk of brain infarction of non diabetic persons.[13]

HYPER LIPIDEMIA

It is one of the risk factor in stroke. Out of 50 patients studied 4 patient had hyperlipidemia. Data clearly support the positive relation between total and LDL cholesterol and a protective influence of HDL cholesterol on extra cranial carotid atherosclerosis. [13]

Transient Ischemic Attack

Incidence of TIA was 12% in our study

Smoking

In our study 10 patients (out of 32 males) were smokers. So the incidence of smoking in males with stroke is 20%. So there is a strong association between smoking and cerebrovascular disease.

Peripheral Vascular Disease

Peripheral vascular disease was observed in 2% of patients. In study conducted by I.R. Stankey, it was amounted to 5%.

Cardiac source of Emboli

Cardiac source of emboli was observed in 10% of cases. In the study conducted by P. M. Dalal the incidence of cardiac source of emboli was 20%.[10]

8% of patient's had atrial fibrillation. In the oxfordshire community stroke project the incidence of atrial fibrillation was 17%.

5 patients had mitral stenosis out of which four were in AF..

Uncertain Causes

Stroke due to undetermined causes was 10% in our study. In oxfordshire community study it was 5%. A definite risk factor was present in 90% of cases in our study t. More than one risk factor was noted in 20% Cases.

Site of Lesion

Middle cerebral artery circulation was involved in 80% of cases.

Vertebrobasilar circulation was involved in 10% of cases.

Anterior cerebral circulation stroke was seen in 10% of cases.

A study conducted by Bomford et al showed middle circulation involvement in 80%, Vertebrobasilar circulation involvement in 15% and anterior cerebral circulation stroke in 5%.

TYPES OF STROKE

Infarct was present in 84% of cases.

Hemorrhagic stroke was 16% according to Harvard cooperative stroke registry study the incidence was 15%.

According to Dalal et al study whose study included 93 cases for a period of 5 years showed.[10]

Ischemic Stroke - 80.60%

Hemorrhagic stroke - 12.50%

When compared with this study our study showed ischemic stroke occurred 3.4% more. And Hemorrhagic stroke occurred 3.5% more.

Among ischemic strokes, thrombosis was 74% and embolism was 26%. In Dalal et al study thrombosis was 70% which is similar to our study.[10]

carotid intima media thickness

In our study, mean carotid intima media thickness in stroke patients was more than control group.

In our study, mean carotid intima media thickness in hypertension was more than control group.

Compared to the normotensive controls, young borderline hypertensive enrolled in the HARVEST showed increased carotid IMT, not only in the common carotid, but also in the bulb and the internal carotid artery.[14]

In our study, mean carotid intima media thickness in L D L, was more than control group. The only lipid parameters which were found to be statistically significant were total cholesterol and LDL cholesterol.[15]

In our study, mean carotid intima media thickness in low H D L, was more than control group. Men with low levels of HDL-C and CHD but without elevated LDL-C or total cholesterol have a very high prevalence of ultrasound-detected carotid artery atherosclerosis.[16]

In our study, mean carotid intima media thickness in hyper glyceride was more than control group. The only lipid parameters which were found to be statistically significant were total cholesterol and LDL cholesterol. Hyperglyceride not significant.[15] Both the group comparisons and the general regression analysis of the pooled data suggest that hypercholesterolemia has an important

role in early onset IMT changes in the common carotid artery, whereas hypertriglyceridemia does not have an appreciable role.[17]

In our study, mean carotid intima media thickness in diabetes mellitus was more than control group. Study provides direct evidence at the vascular level that atherosclerosis levels are elevated before the clinical onset of diabetes. [18]

CONCLUSION

CONCLUSION

The risk factors were present in 90% cases.

The incidence of stroke was common in the age group between 20-60 years.

Among the risk factors hypertension is the most common.

Next to hypertension, smoking, diabetes mellitus, hyperlipidemia, atrial fibrillation , Tuberculous meningitis were the predominant risk factors.

In smokers stroke occurs after an average of 10-20 years of smoking of about 20 cigarette / beedies per day.

Embolic stroke due to cardiac source was more common in age group between 20-40 years with predominance of mitral valvular diseases.

The association of haematocrit / peripheral vascular disease with stroke was less common.

Only about 12% of the patients experienced transient Ischemic attack.

The commonest pathological lesion in stroke was non-hemorrhagic (White) infarction.

The most common vascular supply involved is the middle cerebral artery circulation followed by vertebro basilar artery circulation and anterior cerebral artery circulation,

The predominant type of stroke was ischemic stroke followed by hemorrhagic stroke in that order.

The predominant mode of presentation of stroke was hemi paresis / hemiplegia of acute onset.

For about 10% of the cases no cause could be attributed

In our study, mean carotid intima media thickness increased in stroke than control. Carotid intima thickness increased . In future patient got stroke.

In our study carotid intima media thickness increased in 12% of diabetes mellitus patients with stroke. Carotid intimal media thickness of diabetes increased to develop stroke.

In our study carotid intima media thickness increased in 32% of hypertension patients with stroke, Carotid intimal media thickness of hypertension patients increased to develop stroke.

In our study carotid intima media thickness increased in 8 % low H D L, high L D L, and high triglyceride patients.

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38. **MATHEMATICAL ESTIMATION OF THE POTENTIAL EFFECT OF VASCULAR REMODELLING/DILATATION ON B-MODE ULTRASOUND INTIMA-MEDIAL THICKNESS.** M.L. EIGENBRODT[1], Z. BURSAC[2], E.P. EIGENBRODT[3], D.J. COUPER[4], R.E. TRACY[5] AND J.L. MEHTA

39. **EVALUATION OF CAROTID ATHEROSCLEROSIS BY B'MODE ULTRASONOGRAPHIC STUDY IN HYPERTENSIVE PATIENTS COMPARED WITH**

NORMOTENSIVE PATIENTS M ADAIKKAPPAN, R SAMPATH, AJW FELIX, S SETHUPATHY IND J RADIOL IMAG200212:3:365-368

40. **CAROTID ARTERY INTIMA AND MEDIA THICKNESS AS A RISK FACTOR FOR MYOCARDIAL INFARCTION AND STROKE** NEJM VOLUME 340;1762-1763.

41. **EARLY-ONSET CAROTID ATHEROSCLEROSIS IS ASSOCIATED WITH INCREASED INTIMA-MEDIA THICKNESS AND ELEVATED SERUM LEVELS OF INFLAMMATORY MARKERS** MÁRIA T. MAGYAR, MD; ZITA SZIKSZAI, MS; JÓZSEF BALLA, MD, PHD, DHAS; ATTILA VALIKOVICS, MD, PHD

42. **REPRODUCIBILITY OF ULTRASONOGRAPHICALLY DETERMINED INTIMA-MEDIA THICKNESS IS DEPENDENT ON ARTERIAL WALL THICKNESS THE TROMSØ STUDY** EVA STENSLAND-BUGGE, MD; KAARE H. BØNAA, MD, PHD; ODDMUNDJOAKIMSEN,MD.

43. **INCREASED CAROTID INTIMAL-MEDIAL THICKNESS AND CORONARY CALCIFICATION ARE RELATED IN YOUNG AND MIDDLE-AGED ADULTS THE MUSCATINE STUDY .**PRESENTED IN PART AT THE 70TH SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION, ORLANDO, FLA, NOVEMBER 11, 1997, AND PUBLISHED IN ABSTRACT FORM (*CIRCULATION*. 1997;96)

44. **CAROTID INTIMA-MEDIA THICKNESS IS ONLY WEAKLY CORRELATED WITH THE EXTENT AND SEVERITY OF CORONARY ARTERY DISEASE** MARK R. ADAMS, MBBS, FRACP; AKIHIRO NAKAGOMI, MD; ANTHONY KEECH, MBBS, MEPIDEMIOL, FRACP; JACQUI ROBINSON, RN; ROBYN MCCREDIE, BSC; BRIAN P. BAILEY, MBBS, FRACP; S. BEN FREEDMAN, MBBS, PHD, FRACP; DAVID S. CELERMAJER, MBBS, PHD, FRACP

45. **RELATION OF INTIMA-MEDIA THICKNESS TO ATHEROSCLEROTIC PLAQUES IN CAROTID ARTERIES THE VASCULAR AGING (EVA) STUDY** CLAIRE BONITHON-KOPP; PIERRE-JEAN TOUBOUL; CLAUDINE BERR; CHANTAL LEROUX; FRANCINE MAINARD; DOMINIQUE COURBON; PIERRE DUCIMETIÈRE

46. **THICKENING OF THE CAROTID WALL A MARKER FOR ATHEROSCLEROSIS IN THE ELDERLY**? DANIEL H. O'LEARY, MD; JOSEPH F. POLAK, MD; RICHARD A. KRONMAL, PHD; PETER J. SAVAGE, MD; NEMAT O. BORHANI, MD;

47. **MEASUREMENT OF ARTERIAL WALL THICKNESS AS A SURROGATE MARKER FOR ATHEROSCLEROSIS** ERIC DE GROOT, MD, PHD; G. KEES HOVINGH, MD; ALBERT WIEGMAN, MD; PATRICK DURIEZ, PHD;

48. **LONG-TERM EFFECTS OF LIPOPROTEIN(A) ON CAROTID ATHEROSCLEROSIS IN ELDERLY**

JAPANESE TOSHIHIKO IWAMOTO, SARA FUKUDA, SOUICHIROU SHIMIZU AND MASARU TAKASAKI DEPARTMENT OF GERIATRIC MEDICINE, TOKYO MEDICAL UNIVERSITY HOSPITAL, JAPAN.

49. **CAROTID INTIMA-MEDIA THICKNESS MEASUREMENTS IN INTERVENTION STUDIES DESIGN OPTIONS, PROGRESSION RATES, AND SAMPLE SIZE CONSIDERATIONS:** MICHIEL L. BOTS, MD, PHD; GREGORY W. EVANS, MA; WARD A. RILEY, PHD DIEDERICK E. GROBBEE, MD, PHD

50. **CAROTID INTIMA-MEDIA THICKNESS MEASUREMENTS IN INTERVENTION STUDIES DESIGN OPTIONS, PROGRESSION RATES, AND SAMPLE SIZE CONSIDERATIONS: A POINT OF VIEW** MICHIEL L. BOTS, MD, PHD; GREGORY W. EVANS, MA; WARD A. RILEY, PHD DIEDERICK E. GROBBEE, MD, PHD

51. **CAROTID ARTERY INTIMAMEDIA THICKNESS — A NEW NONINVASIVE GOLD STANDARD FOR ASSESSING THE ANATOMIC EXTENT OF ATHEROSCLEROSIS AND CARDIOVASCULAR RISK?** EVA LONN, MD *CLIN INVEST MED* 1999;22(4):158-60

PROFORMA

Assesement of carotid intimal media thickness in stroke

Name.

Age.

Address.

Occupation

I P No.

Handness

Risk factor

- Hyper tension
- Diabetes mellitus
- Myocardial infarction
- Rheumatic heart disease
- Atrial fibrillation
- Transient ischemic attack
- Previous stroke
- Lipidaemia
- Anemia
- Drug abuse
- Family history
- Connective tissue disorder
- Smoking
- Alcohol
- Sexual contact

History

Onset;

Progress; improvement / worsening

Association symptom

Headache vomiting L. O. C

Fit

Neurological symptom

Hemiplegia hemi sensory loss hemianopia

Speech

Global broca wernicke

Cranial nerve

7^{th} others

Cerebellar involvement

Cardiac status

Periphery vessels status

Carotid

Palpable unilateral bilateral

Laboratory

Blood sugar, random, fasting, post prandial,

Urine examination

Lipid profile

Total cholesterol T G L L D L H D L

Complete hemogram

E C G in all leads

C T scan brain

Carotid Doppler study of common carotid artery

And internal carotid artery. .

Echo cardiogram.

Blood V D R L.

ANNEXURES

ABBREVATION

T I A – Transient ischemic attack.

D M – Diabetes mellitus **.**

SHT- Systemic hypertension.

H D L – High density lipoprotein.

L D L- Low density lipoprotein.

T G L- Triglyceride.

CAROTID A I M T- Carotid artery intima media thickness.

C C A. Common carotid artery.

I C A - Internal carotid artery

HEMO-Hemorrhagic

THROMBUS- Thrombotic

M Male

F Female

S. NO	NAME	AGE	SEX	DIAGNOSIS	TIA	SMOKE	DM	SHT	HDL	LDL	TGL	CAROTID. A. I M T	
												CCA	ICA
1.	Rajammal	35	F	thrombus	No	No	No	yes	60	150	160	0.66	0.68
2.	Kamalamal	42	F	thrombus	yes	No	No	No	60	140	150	0.68	0.67
3.	selvakumar	28	M	thrombus	No	No	No	No	55	130	160	0.77	0.74
4.	planiammal	57	F	hemo	No	No	No	No	50	150	175	0.68	0.67
5.	Mani	52	M	thrombus	No	No	No	No	55	150	160	0.92	0.91
6.	nanjappan	48	M	thrombus	No	No	Yes	yes	40	180	250	0.88	0.87
7.	Raman	37	M	thrombus	No	Yes	No	yes	55	150	160	0.69	0.68
8.	Robesa	30	F	Hemo	No	No	No	No	60	140	180	0.67	0.65
9.	chinnan	47	M	thrombus	No	No	No	No	55	110	140	0.77	0.74
10.	valliammal	35	F	thrombus	No	No	No	No	65	100	130	0.65	0.67
11.	rajagopal	48	M	Hemo	yes	yes	yes	No	35	185	220	0.68	0.67
12.	ramasamy	59	M	thrombus	No	yes	No	yes	55	160	170	0.64	0.62
13.	chokkan	62	M	thrombus	No	No	No	No	60	150	150	0.68	0.64
14.	Kitten	35	M	thrombus	No	No	No	No	55	160	170	0.63	0.68
15.	mannan	51	M	thrombus	No	yes	No	yes	60	130	150	0.67	0.64
16.	lakshmi	46	F	thrombus	No	No	No	No	55	150	160	0.67	0.61

S. NO	NAME	AGE	SEX	DIAGNOSIS	TIA	SMOKE	DM	SHT	HDL	LDL	TGL	CAROTID. A. I M T	
												CCA	ICA
17.	Rajathi	49	F	thrombus	No	No	No	No	65	130	150	0.62	0.67
18.	Rangan	65	M	Hemo	No	No	No	No	64	120	130	0.63	0.65
19.	Palani	43	M	thrombus	No	No	No	yes	55	160	170	0.67	0.68
20	Murugan	75	M	thrombus	No	yes	yes	No	30	185	220	0.69	0.68
21.	Chinnasamy	64	M	thrombus	No	No	No	yes	55	160	150	0.77	0.79
22.	Mohideen Beebi	51	F	thrombus	yes	No	No	No	60	130	160	0.78	0.77
23.	Saleema	49	F	thrombus	No	No	No	yes	60	150	170	0.79	0.76
24.	Kurnal	65	M	thrombus	No	yes	No	No	55	150	160	0.77	0.77
25.	Akbar ali	51	M	thrombus	No	yes	No	yes	60	130	150	0.78	0.85
26.	Karuppammal	48	F	Hemo	No	No	No	No	65	150	160	0.92	0.93
27.	Omana	69	F	thrombus	No	No	No	No	55	130	155	0.84	0.84
28.	Palaniappan	53	M	thrombus	yes	No	No	No	50	140	160	0.78	0.77
29.	Manikandan	61	M	thrombus	No	No	No	yes	60	150	160	0.78	0.77
30.	Sanker	49	M	Thrombus	No	No	yes	No	40	180	220	0.77	0.76
31.	Chellappan	39	M	Hemo	No	No	No	No	60	150	170	0.75	0.77
32.	shantha	62	F	thrombus	No	No	No	No	55	150	170	0.87	0.85

S. NO	NAME	AGE	SEX	DIAGNOSIS	TIA	SMOKE	DM	SHT	HDL	LDL	TGL	CAROTID. A. I M T	
												CCA	ICA
33.	Shanmugan	51	M	thrombus	No	yes	No	yes	60	140	150	0.77	0.71
34.	Dharaman	35	M	thrombus	No	No	No	No	55	130	150	0.75	0.74
35	Indhu	66	F	thrombus	No	No	No	No	60	140	160	0.64	0.65
36.	mohamed	49	M	thrombus	No	No	yes	No	40	170	170	0.67	0.68
37.	suresh	61	M	thrombus	No	No	No	No	60	150	150	0.69	0.69
38.	sathis	43	M	thrombus	No	No	No	No	55	130	140	0.77	0.75
39.	indrani	59	F	thrombus	No	No	No	yes	60	110	130	0.68	0.69
40.	sheela	52	F	thrombus	yes	No	No	No	55	130	150	0.64	0.65
41.	Kanagaraj	57	M	Hemo	No	No	No	No	60	140	170	0.55	0.54
42.	Rama krishnan	55	M	Thrombus	No	yes	yes	yes	35	185	170	0.64	0.65
43.	Thirumal	67	M	Thrombus	No	No	No	No	60	120	150	0.62	0.67
44.	shobhana	64	F	Thrombus	No	No	No	No	55	130	150	0.67	0.61
45.	Vargeese	57	M	Thrombus	No	yes	No	yes	60	110	130	0.67	0.68
46.	eswaran	61	M	thrombus	No	No	No	No	55	130	150	0.64	0.67
47.	Meenashi	54	F	Hemo	No	No	No	No	55	110	140	0.67	0.63
48.	lakshmi	67	F	thrombus	yes	No	No	yes	60	100	130	0.62	0.66
49.	Moorthy	58	M	thrombus	No	yes	No	No	65	110	140	0.63	0.61
50.	Devi	52	F	thrombus	No	No	No	yes	55	130	150	0.66	0.65